LETTERS TO THE EDITOR

Labial adhesions following severe primary genital herpes

EDITOR,—Labial adhesions following genital herpes infection have been described previously.[1–4] To prevent their development various suggestions such as the use of early aciclovir,[1] paraffin gauze,[2] and saline bathing[3] have been put forward. We believe nursing care is a significant factor in the prevention of this complication. Here we report two cases of severe genital herpes presenting at different sites, almost at the same time, both necessitating admission and developing labial adhesions.

CASE 1

A 25 year old woman was admitted to the medical ward with severe vulval ulceration, generalised skin rash, and difficulty in micturation of 4 days' duration. Clinical examination revealed target lesions, swollen labia, bilaterally enlarged tender inguinal lymphadenopathy with extensive vulval ulcerations. A clinical diagnosis of erythema multiforme secondary to herpes simplex virus (HSV) was made. However, swabs taken at admission for HSV culture were negative. The patient was commenced on oral aciclovir and metronidazole and advised to use topical lignocaine gel; she admitted, however, to being afraid to touch her genitalia. The patient made a slow recovery and was allowed home following 8 days in hospital. At follow up (GUM) 2 weeks later, she presented with a history of her abnormal urinary stream "urine splashing all over the place." Examination of the external genitalia revealed two bands of adhesions between the labia minorae. The bands were separated using a knife after infiltration with lignocaine 2% and gauze dressing dispensed to prevent further adhesions. No clinical abnormality was detected on follow up.

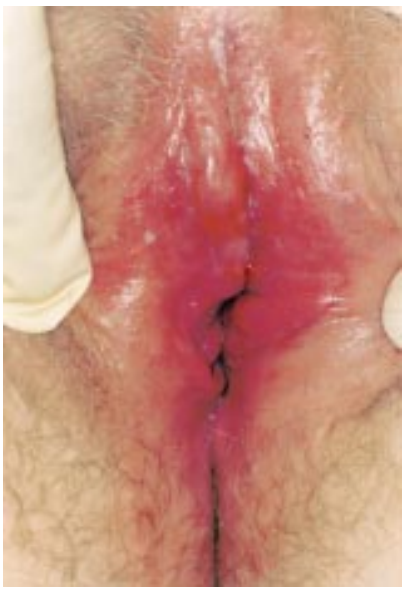

Figure 1 (Case 2). Thick band of adhesions between the middle halves of labia minorae.

CASE 2

A 27 year old insulin dependent female diabetic was admitted to the gynaecology ward with history of acute onset of vulval soreness, fever, and difficulty in micturation of 3 days' duration. On examination she had a temperature of 38.2°C, oedematous tender vulva, and bilaterally enlarged tender inguinal lymph nodes. A presumptive diagnosis of cellulitis was made. The patient was catheterised and commenced on topical lignocaine gel, subcutaneous morphine, intravenous metronidazole, and cefuroxime, and insulin by sliding scale. Two days later she developed perineal and vulval ulcerations and intravenous aciclovir was added. In view of failure of clinical response the genitourinary department was asked to review the case. Examination revealed perineal and perianal ulcers. A diagnosis of primary HSV was made, intravenous antibiotics were stopped, and oral antivirals were started. The nursing staff were instructed to offer the patient a Sitz bath twice daily in view of extensive discomfort and oedema. Swabs taken confirmed the diagnosis of HSV. The patient made a gradual recovery and she was allowed home after 1 week in hospital. Two weeks later when she presented to the genitourinary medicine clinic, genital examination showed a thick band of adhesions between the middle halves of the labia minorae, and new herpetic lesions (fig 1). She was prescribed oral valciclovir, metronidazole, and lignocaine gel and advised to continue salt and water bathing at home. A follow up appointment was arranged for release of adhesions. Surprisingly, separation of adhesions was not needed.

COMMENT

These two cases illustrate that females with severe genital herpes can be admitted to different hospital departments other than genitourinary medicine, where the nursing staff may not be familiar with the management and complications of this infection. Patients should be encouraged to separate the labial folds; this can be facilitated by the liberal use of local anaesthetic agents with the assistance of the nursing staff. Frequent saline bathing of the genitalia should be encouraged to facilitate the removal of the fibrinous exudate, which is responsible for the formation of these adhesions.

GUM nurses and physicians should play an active part in the education and nursing care of such cases and lead the management especially when admitted to other specialties.

Contributors: EH managed case 1, JD managed case 2, while both authors wrote the manuscript.

E HERIEKA
Department of Genitourinary Medicine, Leicester Royal Infirmary

J DHAR
Department of Genitourinary Medicine, Derbyshire Royal Infirmary

Correspondence to: E Herieka, Department of GUM Leicester Royal Infirmary, Leicester LE1 5WW, UK

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Respiratory and cutaneous manifestations of disseminated cryptococcosis in AIDS

EDITOR,—A 26 year old, previously fit and well Afro-Caribbean man, presented with a 5 week history of a "flu-like" illness. Initially treated with antibiotics, the patient deteriorated, developing a cough, haemoptysis, progressive breathlessness, intermittent blurring of vision, and a rash. Investigations indicated he was HIV positive.

On examination, though orientated, he looked unwell and was febrile. He had an extensive papulonodular rash on his face, trunk, and limbs. Many of these lesions were centrally umbilicated with areas of associated haemorrhage (fig 1). Respiratory examination revealed decreased air entry in the right chest and coarse inspiratory bi-basal crackles. Funduscopy demonstrated retinal pallor, congested optic discs, and bilateral soft exudates associated with haemorrhages. He had no focal neurological signs.

Full blood count, urea and electrolytes, and clotting screen were normal. Arterial blood gases on 35% oxygen revealed a pH of 7.44, Pa_{O_2} 9.4 kPa, Pa_{CO_2} 2.7 kPa, base excess −8.2. Chest radiograph demonstrated bilateral infiltrates with a right sided pleural effusion.

The patient had been treated for a presumed diagnosis of severe community acquired pneumonia and/or *Pneumocystis carinii* pneumonia plus *Molluscum contagiosum* of the skin. In view of the patient's clinical findings, additional therapy was commenced with anticytomegalovirus (CMV) and anticryptococcal agents.

Urgent blood and pleural fluid cryptococcal reactive antigen testing (CRAG) were strongly positive at a titre of >1:2048. Blood CMV PCR was negative. The patient could not tolerate a lumbar puncture. Despite initial improvement, he developed progressive respiratory failure and died. The post mortem revealed disseminated cryptococcal disease with involvement of brain, skin, lung, heart, liver, spleen, kidneys, pancreas, thyroid, bowel, adrenal glands, and testes.

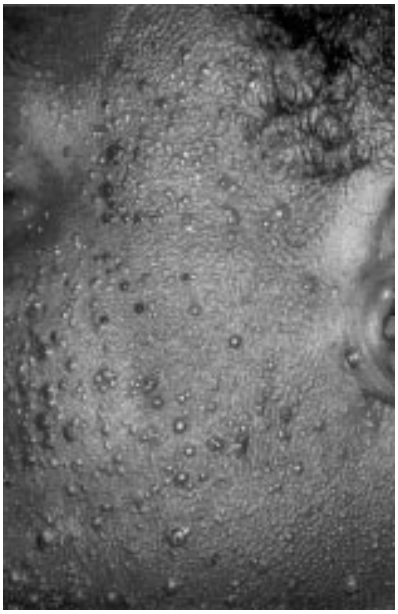

Figure 1 Cryptococcal skin lesions associated with disseminated disease.